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DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

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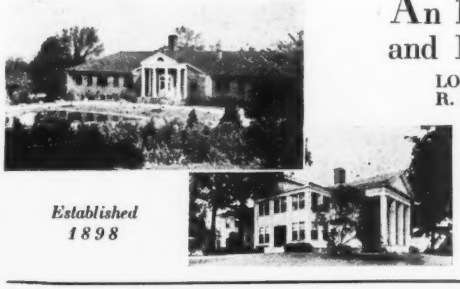

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DISEASES of the NERVOUS SYSTEM

(PUBLISHED MONTHLY)

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

EDITORIAL COMMENT

This, the December 1941 number of *Diseases of the Nervous System*, marks the completion of its second successful year of publication. In starting a journal of this kind the editors and associate editors went into the question of publishing this type of journal with some degree of uncertainty; but after due consideration and discussion with a number of physicians throughout the country, it was felt very definitely that it could be made a success and that there was a very definite place for this type of publication. There is no question but what psychiatry and mental hygiene, and particularly neurosurgery, are occupying an increasingly important part in the whole field of medicine, and, therefore, it is necessary that every physician make himself acquainted with the various phases of neurology and psychiatry. It was the realization of this fact that created the idea of publishing this journal, and we are happy to have had it pointed out by neurologists, psychiatrists, and physicians in general practice as well, that this journal is fulfilling this purpose.

We are highly pleased with the way the journal has been received, and the editors wish to take this opportunity to express their appreciation to all of those who have contributed articles during the past two years; it is our earnest hope that they will continue to send in papers for publication. As in the past, it is our wish to publish short articles dealing more with the practical phases of the diagnosis and treatment of the various neuropsychiatric conditions, rather than the more scientific articles of research interest, feeling that the former type of articles would be of more value to the general practitioner, as well as the fact that other excellent journals on neurology and psychiatry are available for the latter type of papers.

We regret that it is necessary at times to hold very splendid articles for a long period of time before they can be published, but at the present time, when we are unable to publish more than five or six articles in each issue, it is necessary to delay publication, particularly since there are such a large number of good papers on hand. Perhaps in the near future the journal will be enlarged so that we can publish more contributions in each number.

We are pleased to announce, too, that the circulation of the journal has increased considerably, and more and more hospitals are taking advantage of the journal as an advertising medium.

We are looking forward to an increasingly successful year for 1942.

T. H. H.

Alcoholism: A Public Health Problem

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Boston, Massachusetts

Dr. Moore was graduated from Vanderbilt University School of Medicine in 1924. He is an Associate in Psychiatry, Department of Diseases of the Nervous System, Harvard Medical School, Visiting Psychiatrist, Neurological Unit of the Boston City Hospital and Medical Director of the Washington Hospital, Boston. Dr. Moore is a Member of the American Neurological Association, the American Psychiatric Association, the Massachusetts Psychiatric Society, the New England Psychiatric Society and the Boston Society for Psychiatry and Neurology.

There are few problems which touch the lives of as many persons, either directly or incidentally, as alcoholism. It has important connections with public health and has sociological, psychological and economic implications of great significance, in terms of expense as well as human suffering.

Alcoholism is of minor importance as a principal cause of death; but as a contributory factor, favoring the development of organic disease, it ranks high. However, its part as a contributing factor, although recognized, is poorly defined, since no real control studies are possible. Its cost to the individual, the family and the community cannot be expressed in simple figures, for it involves such intangible and remote costs as the tax rate on your real property, the amount of money available in a tax dollar for schools and roads, and the dividends which invested funds yield. When one reckons the cost of caring for chronic alcoholics in jails and for acute alcoholics in hospitals, the resulting figures represent only the surface cost. Persons with alcoholic psychoses increase the budget of the Massachusetts State Department of Mental Health by 10 per cent; broken homes resulting from alcoholism cause municipal and federal budgets for relief to be greatly increased; the costs incident to accidental deaths which are indirectly due to the intemperate use of alcohol are tremendous. In a general sense, the term alcoholism covers the acute and chronic condition as well as the alcoholic psychoses and in this paper the term generally is used to refer to the entire social, psychological, biochemical and medical problem. If one attempts to define alcoholism, it can be described as the condition that occurs when a person drinks to an extent that interferes with his work or home life.

Ethyl alcohol has been used by man since antiquity in the pursuit of pleasure (by well-adjusted people) and as a form of escape by others who are either physiologically unable to handle it well or who, psychologically, seem to possess low resistance to its effect and seek euphoria and anaesthesia. The majority of well-adjusted, essentially normal people who drink never come to harm, and for one individual who does suffer from its excessive use, there are probably twenty who are unaffected by it and who drink in moderation. Its use is a problem which only the individual can solve for himself.

The scientific attitude aims only at the collection and application of factual data and holds no brief for the moralistic and regulative attitude, nor for the commercial attitude of individualism which denies responsibility after manufacture and sale have been completed. Happily, the groups which hold these two essentially emotional attitudes are compromising their stand somewhat, and representatives of the commercial, the scientific and moralistic groups are finding a common meeting place today through the formation of the Research Council for the Problems of Alcohol. This is an organization which seeks through the medium of the Quarterly Journal on Problems of Alcohol, now completing its first half-year, and through other avenues to distribute information about alcohol. It is hoped that educational and therapeutic methods may serve to bring about the end in which the Eighteenth Amendment failed so miserably, i.e., the rational use of alcohol and the prevention of alcoholism. The scientific attitude is in no wise interested in personal profits, but is no less practical for that. The work of the Research Council is growing rapidly. In December, 1940, it presented a three-day program to the American Association for the Advancement of Science in Philadelphia. At this meeting the outstanding authorities in America gave a comprehensive summary of the entire field. Representatives of the courts, social service, medicine, chemistry, the distilling industry, and the clergy participated in this symposium.

The fate of alcohol in the body is well understood. About 95 per cent of it is oxidized to yield 7 calories per gram and the balance is excreted via the kidneys, lung and skin. The onset of intoxication varies with the individual, but when the blood alcohol concentration reaches 150 milligrams per hundred cubic centimeters, there are unmistakable signs of intoxication. At a level of 250 milligrams, few persons can walk without staggering, and when the blood alcohol concentration reaches 400 milligrams, coma begins. Recovery is rare if the blood level reaches 500 milligrams or 0.5 per cent. Of course, it must be remembered that the process of oxidation proceeds at a uniform rate, and some is disappearing from the blood as more reaches it from the gastro-intestinal system. Therefore it is necessary to drink a very large volume in a very short period to reach fatal concentrations in the blood. For the average man, this volume would be more than a pint of pure alcohol in less than an hour. Tolerance and habituation change the time relation of the blood alcohol curve somewhat, although these factors are still somewhat debatable. They seem to bear an intangible relation to personality but this is, at present, a hypothesis, an impression rather than a proven fact. It may be only a chance correlation that an individual with a well-organized personality, who finds no difficulty in integrating his behavior, is apparently unaffected by a quantity of alcohol which will raise his blood alcohol level to 100 milligrams, while the same amount may cause a marked change in a person who is hysterical or neurotic in his makeup (and badly adjusted socially). Physicians are still considering whether there may be a common physical basis underlying the general personality structure and the tolerance to alcohol. Nevertheless, many patients are seen in psychiatric practice who simply cannot drink the smallest amount of alcohol with impunity.

The diagnosis of acute alcoholism is fairly simple. There have been recommended a number of chemical procedures: but, for the most part, these cannot be carried out without the assistance of a trained chemical worker. They require the drawing of a sample of blood and in medical-legal cases this cannot always be obtained at a given time. Delay may mean that the blood alcohol concentration has dropped significantly. In one American city, movies have been made of persons arrested for alcoholism which constitute the record of the accused prisoner's behavior. There is considerable doubt as to the exact constitutional status of such evidence, since no man can be forced to bear witness against himself.

Dr. Walter Jetter[1] of the Taunton State Hospital has devised a simple apparatus for examining a specimen of expired air by allowing it to pass through a quantity of magnesium perchlorate which absorbs the alcohol somewhat selectively. The tube can be stoppered and analyzed later. In a previous series of control experiments, Dr. Jetter worked out the relation of carbon dioxide and alcohol in the expired air to that in the blood, and he now can compute the blood alcohol content accurately from his results. This technique is a modification of earlier methods, such as that devised by Harger and Hulpieu in Indiana, but it is believed to have certain practical advantages over theirs. The status of this examination in the courts has not been defined, but it has been commended by a number of medical-legal authorities and may some day be acceptable as evidence.

The diagnosis of chronic alcoholism is much more difficult, for when a man drinks and is not intoxicated, yet displays the abnormal behavior of chronic alcoholism, the problem is quantitative rather than qualitative. We no longer ask ourselves after a psychiatric examination, "Has this patient been drinking?", but rather, "To what extent is this man's drinking responsible for his abnormal behavior?" To increase the psychiatrist's dilemma, it is almost universally true that chronic alcoholism is merely an expression of underlying psychiatric abnormalities, and when alcohol is withheld some other means of anti-social conduct usually is employed as an outlet for the basic drives which constitute the primary disturbance.

The pathology of alcoholism is not definite and many of the lesions traditionally attributed to prolonged use of alcohol have not been produced in experimental animals. Dr. Frank B. Mallory and Dr. Timothy Leary have tried for many years to produce cirrhosis of the liver with alcohol without success. The chronic alcoholic may have gastro-intestinal lesions as a result of alcoholic excess, but this not a constant finding. Dr. Robert Fleming[2] has

pointed out that the morning vomiting of thick, glairy mucous material so often seen in the chronic alcoholic constitutes the only consistent physical symptom encountered in this condition. It is the only symptom found in almost all cases of chronic alcoholism. It is probably true that a lifetime of alcoholic saturation does not favor the maintenance of sound blood vessel walls nor exert an effect like digitalis on the myocardium, but so far no one has been able to produce significant organic impairment of the cardio-renal system in animals, although their reaction to acute alcoholism is in no essential particularly different from that of man.

There is, however, one type of disease which occurs in the chronic alcoholic with great frequency, but it is not directly due to the pharmacological effects of alcohol. These are the avitaminoses.[3] The life of the usual chronic alcoholic addict or of the periodic drinker (the type that indulges in "sprees") is characterized by a grossly inadequate diet either from the standpoint of nutritional intake or balance.

The alcoholic tends to eat very irregularly, if at all, and when he does, his diet consists of high carbohydrate items such as bread, crackers, macaroni, or potatoes. This is in many cases due to the limited money at his disposal. But in distributing his expenditures, if he thinks about it, he reserves the bulk of his budget for liquor. In the case of chronic drinkers who are not confronted by an economic problem, the foods of choice seem to be those with exceptionally low vitamin and mineral content. Thus, over a period of time, the vitamin reserves are depleted and may never be replenished. It does not require a long period of time for this to occur, for we have seen cases at the Boston City Hospital, and they have been reported from the Bellevue Hospital, in which the signs of acute vitamin deficiency have appeared within four weeks after the beginning of dietary imbalance. These conditions include peripheral neuritis, polyneuritis, toxic neuritis, central neuronitis, and Wernicke's disease.

Since it has been possible to quantitate the ascorbic acid content, it has been shown that the chronic alcoholic has a subnormal vitamin C level and in many cases a subclinical scurvy has been diagnosed by this examination. In a study made by Alexander, Pijoan, Schube and Moore[4] at the Boston State Hospital several years ago, it was found that patients with alcoholic psychoses had a lower blood plasma content of ascorbic acid than did control patients. The ascorbic acid content in these alcoholic patients was often as low as in scurvy. In view of the fact that some of these patients had been in hospital residence for many years, and considering some other evidence[5] regarding the oxidation of alcohol by the chronic alcoholic, it can be hypothesized that such patients do not have a cellular oxidative mechanism which is functionally as effective (with relation to alcohol) as do non-alcoholic individuals. Of course, factors other than diet may lower the ascorbic acid content, such as infection, but the patients selected for our study were for the most part in good physical health.

The lowering of his vitamin reserves may predispose an individual to a subdural hemorrhage, although the exact relation between the two conditions is not known. In a study of patients who were found at autopsy to have subdural hemorrhages (many previously undiagnosed) the factor of alcoholism loomed large, although some of them had been hospitalized for long periods before death.

The records of 3100 autopsies performed consecutively in Massachusetts State mental hospitals[6] showed that 7.9 per cent of the patients in this series had subdural hemorrhage. Althaus[7] recognized the relation between subdural hemorrhage and alcoholism as early as 1878, when he observed that the former condition occurred in "old, decrepit and intemperate" persons. In the series studied in Massachusetts State hospitals, there was an incidence of 83 alcoholic cases per 1000 cases of subdural hemorrhage. No case was under 30 years of age. In these patients, numerous personality changes had been observed.

The principal cause of death in alcoholic patients is seldom alcohol. Other factors are almost always present as complications. The uncomplicated cases account at the Boston City Hospital for less than one per cent of deaths among alcoholics. In a ten-year study[8] of deaths in Massachusetts due to poisons, the records of all deaths certified by medical examiners were reviewed in the office of the Secretary of State. Deaths due to alcohol constituted 52 per cent. Although this figure

appears large, when it is related to all deaths in Massachusetts from all causes during that period, it is proportionately small. However, it should be pointed out that the causes of deaths as recorded on death certificates are the subject of considerable confusion. There are a number of Standard Classifications of Disease and causes of death. The most useful in my experience is that presented by the Bureau of the Census in a pamphlet, "Physician's Handbook on Birth and Death Registration." It is quite probable that as a cause of death, alcohol is more important than we can demonstrate by the use of statistics, for it is a common and questionably humane custom to spare the relatives of a drunkard the ignominy of such a final diagnosis. This practice is unfortunate from the scientist's and the statistician's point of view.

In the study of deaths from poison in Massachusetts from 1928-1937, it was found that a total of 88,090 cases had been certified by medical examiners, and that 8,861 of these were due to poisonous substances, representing 9.83 per cent of all deaths. 4,505 of those deaths from poisoning (or 5.11 per cent of all deaths) were due to ethyl alcohol. If the cases of fatal accident or suicide while intoxicated were added to them the number reached 4,742 or 5.38 per cent of all deaths. Thus alcohol not only causes more deaths than any other poison, but it causes more deaths than all other poisons put together. Yet, when related to the total number of deaths from all causes, this figure is not excessively large. In the Bureau of Census table for 1937,[9] among the ten principal causes of death (the so-called "killers") alcohol and drugs are not included, yet the ten principal causes cover 72 per cent of all deaths for that year, for the entire country.

As a general rule, alcoholics are not prone to suicide, and when they do attempt it they are not especially successful. This may be due to the fact that alcohol may release for some individuals the same self-destructive urges that others express in acts of physical violence, such as shooting, slashing or taking other poisons by mouth, and its results are less permanent. If it be a method of self-destruction, we cannot demonstrate that it is a conscious form, and assuming that it may be consciously used for suicidal purposes in a few instances, it is about as ineffectual as iodine.

Among 1147 cases of attempted suicide at the Boston City Hospital, [10] 143 were alcoholic at the time of attempt and only seven (or 5 per cent) of these were ultimately successful. The average age of both men and women at the time of their attempt was between 30 and 40 years, slightly older than the average for all alcoholic patients, but well within the age group of most of the hospitalized acute and chronic alcoholic patients.

The histories of these patients show the breaking of personal adjustments and continued symptomatic drinking often in a periodic pattern, or addiction, accompanied by episodes of emotional crisis sometimes preceded by depression, and bewilderment. When sober and recovered, these patients were embarrassed and discouraged and were still confronted and disturbed by various psychological and social difficulties. Many of them were not frankly psychotic, but some may have had pre-psychotic personalities or may have suffered from constitutional psychopathic inferiority or hysteria. Most of them were of low economic status, but that is true of most patients seen at the Boston City Hospital. We do not have similar data regarding successful alcoholic suicides, but there is little to suggest that they are, as a group, very different from the non-successful ones.

Considerable information on alcoholism as a hospital problem in Boston is available. The data available for this community has been compared with that of investigators elsewhere in the country and it appears that the situation, with respect to hospitalized alcoholics, is fairly typical. In the first seventy years of the Boston City Hospital about 50,000 alcoholic patients were treated, or 5 per cent of all patients admitted.[11] Yet according to the legal provisions under which the hospital was established, no persons was eligible for treatment unless he or she suffered from a medical or surgical condition.

In his address at the dedication of the Boston City Hospital in 1865, Mayor Amory remarked: ". . . But the respectable poor, virtuous, neat and well-conducted should not be subjected to the daily intercourses with the profligate, who by intemperance and viscious indulgences have degraded themselves to the level of the brute; whose unseemly

habits, profane and rude conversation, would soon drive all others away. For their convenience these apartments were never intended, but both City and State have made other provisions. The government should be firm in declining to receive them, in compelling their removal when inadvertently admitted."

Apparently this prudent counsel was found difficult of application, for references to the same problem continue to recur in later Annual Reports. That for 1879, for example, remarks: "But . . . there are other classes of people who are . . . victims of their own vices with alcoholism or injuries from drunken quarrels or accidents, who infest the Hospital and whose conditions at the time of application makes it inhumane to raise any question as to their admission. For all such, certainly there can be no doubt that the City should be repaid under the law, for the cost of their care." There is, however, no record to date that repayment was made.[12]

A distinct change is observable in the policy of the Hospital in 1879, for the Annual Report states: "The policy of its (the Hospital's) management, as the Trustees are aware, has been the definite one of endeavoring to meet in every way practicable the wants of the public, for whom the Hospital exists, by the admission of all persons who have any claim upon it for medical aid and treatment." After the establishment of the Haymarket Square Relief Station in Boston the same policy was followed theoretically, but in practice it was never too successful because most of the alcoholics treated there suffered also from complications, as we shall see presently.

There was a steady increase in alcoholic admissions at the main hospital from 1 to 14 per cent and the average number was 7 per cent for the 70-year period. Of course, there was also an increase in all admissions, but the increase in alcoholic patients was usually great as the policy of the Hospital became progressively relaxed toward them. Probably during Prohibition years, the number was highest but after repeal it did not fall off to any extent. Male patients have outnumbered female patients in the ratio of 7 to 1, but this may be due to the fact that women are often cared for at home, if alcoholics, where they might otherwise be hospitalized if the pride and shame of the family did not intervene. The average age of the males was 36 to 40 years and that of females was 41 to 45 years. Eighty-four per cent were discharged as relieved, but many of these repeated their hospital entry many times. Those who died in the hospital usually entered in coma and succumbed soon after admission.

On admission, many of these patients were found to be suffering from neurological diseases, especially the neuritides and deficiency diseases such as scurvy and pellagra. Delirium tremens was a frequent occurrence. I do not have at hand the data for the incidence of delirium tremens over the entire 70-year period mentioned above, but for the twenty years between 1915 and 1935, 38,376 patients were admitted because of alcoholism and 2375 of these were diagnosed delirium tremens.[13] This figure represents an annual average of about 250 cases, or 6.2 per cent, of all alcoholics. In 1930 there were 3 per cent, but in 1935 delirium tremens cases constituted 11 per cent of all alcoholics. There is a high rate of fatal outcome when delirium tremens occurs. In the 20-year period, 560 patients with delirium tremens died, but the percentage of death had dropped from 52 per cent in 1915 to only 14 per cent in 1935. In 1932 only 7 per cent of delirium tremens patients in the Boston City Hospital died. The lowering of the death rate in this group seems to be related to better understanding of the condition and to improved methods of medical treatment and nursing care. Men predominate among fatal cases, and the average age at admission for all male cases of delirium tremens is 36-40 years. For women the average age is 41-45 years.

An evaluation of the causes of death in cases of delirium tremens is difficult. However, two groups seem to stand out: those in which the cause of death is directly related to alcoholism, and those in which the relation is only indirect or coincidental.[14] Pneumonia is a common complication of delirium tremens. Studies by Weichselbaum, Grawitz and others indicate that alcoholism and the associated vitamin deficiencies cause a diminution in resistance to bacterial infections. Cases with dilation of the heart ("Bier-herzen") are most frequently found in the vitamin deficiency states associated with alcoholism. These have been proved to be similar to beriberi heart disease by the studies of Weiss and

Wilkins. A large number of alcoholic patients die from direct involvement of the brain due either to neuronitis, pellagra or to Wernicke's disease, which conditions may involve the vital centers of the brain, including the vagus region. Wernicke's disease may be recognized grossly, particularly if the lesions are associated with hemorrhages, but these cannot be observed in all cases. In the absence of visible hemorrhage, an experienced pathologist may recognize the bluish-gray discoloration of the periventricular tissue as suggestive of Wernicke's disease. A neuronitis of the pellagra or the beriberi type can be recognized only microscopically. Advanced neuronitis of the vagus nucleus should be accepted as a cause of death, as pointed out by Alexander.

In studying the situation at the Haymarket Square Relief Station in Boston, these points became obvious: alcoholic cases which were in need of treatment beyond that which could be given in one day were usually transferred to the main hospital. Male alcoholic patients at the Haymarket Square Relief Station outnumbered females in the ratio of 18 to 1 between 1927 and 1937. Alcoholics constituted 33.6 per cent of all male house patients and 13.5 per cent of all female house patients. Actually, alcoholics made up only 3.4 per cent of all patients because the large majority of patients treated at this institution were suffering from minor injuries and were attended in the Out-Patient Department. The average stay of the alcoholic house patient was one day and the maximum stay (in one case) was 26 days.

Interviews with these patients revealed occupational maladjustment, domestic friction, loneliness and frustration to be the chief reasons they gave for drinking. In the majority of cases, basic underlying personality defects were present, as well as feelings of inferiority which were poorly concealed by the alcoholic episodes.

Although the total number of alcoholic patients represented only 3 per cent of all patients, the cost of their care was 6 per cent of the total hospital budget. This was estimated by multiplying the per diem cost by the number of days of hospital stay. In a group of alcoholic patients treated at the Haymarket Square Relief Station during the 15-year period from 1923-1938 (15,229 men and 825 women), 94.9 per cent were men and the average age was 40-45 years at the time of admission. Fifty-eight per cent of the male patients and 25 per cent of the women were unmarried. Most of them were born in Massachusetts, but of the foreign born group, persons born in Ireland, Canada and the British Isles predominated in that order. The racial predominances probably reflect nothing more than the percentage of these nationality groups in the neighborhood. Thirty-two countries were represented as birthplaces. Two-thirds of the patients lived in Boston, many near the hospital, and were for the most part unskilled workers. 12,712 were brought in by the police.

Alcoholism was not always the most prominent symptom, but each patient had evidence about his person of recent drinking. On discharge, most of them were relieved and were discharged unconditionally, but a large number were returned to the police or courts. The chief complications were surgical, but pneumonia, malnutrition, gastro - intestinal disturbances and neurological disorders were common.[15] Many were suffering from frank gastritis and neuritis, cerebral vascular conditions and some patients were having convulsions or "rum fits" when admitted. The most common surgical complications were fractures and head injuries, the latter numbering 10,494 in men and 380 in women out of a total of 16,054 injuries varying in severity from abrasions to compound fractures. Injuries to the arms and legs were next in number and included lacerations, abrasions, contusions and abrasions, contusions and fractures, in that order. There were frequent cases requiring treatment for epistaxis, concussion, immersion, dislocations, and ecchymoses. Of the medical complications, 517 were neurological and 240 were gastro-intestinal. 138 were cardiovascular and 101 resulted from poverty, malnutrition or exposure. The other body systems were affected, but less frequently. The distribution of medical complications was not very different than might be found among non-alcoholics of the same economic level.

The causes of death among these patients deserve brief comment, although they numbered only 75 in 15 years (due to the practice of removing serious cases to the main hospital without delay).[16] Only 3 women died at the Haymarket Square Relief Station. All but

7 cases died within 24 hours of admission. Head injuries caused death in 17 cases, and fractured skulls in 15. Cardiac deaths numbered 15. Other causes were concussion, pneumonia, cerebral edema, delirium tremens, epistaxis, malnutrition and starvation, dislocation of the jaw, bullet wounds, burns, veronal poisoning (suicidal), pulmonary hemorrhage and ruptured liver. There were a few cases in each of these categories.

There was little correlation between the age of these alcoholic patients and their intelligence.[17] It might be said generally that they were low normal and that their education was limited. The older men were chiefly laborers and seamen and the men of middle age were usually salesmen and longshoremen. In many cases there had been a failure to make a satisfactory occupational adjustment and a decline with increasing age into unskilled labor groups. Social and economic factors, foreign birth and language difficulty may have also been contributing factors in their decline.

These statistics have been presented at some length in order to show alcoholism as a public health problem which is fundamentally cotangent with many other fields and to show its proportions. In the minds of many persons there are questions about the treatment of alcoholism and its effectiveness. For the acutely intoxicated person, sedation, the restoration of a normal acid-base and water balance and supportive measures are indicated. These are well understood by most physicians.

For the chronic alcoholic addict, only psychotherapy seems to have much value. When there is marked deterioration of personality and the development of psychotic behavior, the patient should be examined by a psychiatrist if possible. In many circumstances this is impossible and psychiatric handling of the case must be undertaken by the general physician.[18] Before psychotherapy is begun, it is essential that the patient should have become free of alcohol following such symptomatic measures as will calm him, enable him to rest, encourage normal elimination and permit him to regain normal fluid balance. The recent memory of the unpleasant symptoms associated with intoxication may be adequate to restrain the patient from drinking for a few days, but memory soon dims, and unless a new program of activity is outlined, the cycle of drinking is begun again. The importance of physical well-being as a fundamental step in promoting mental stability is one of the most basic truths in psychiatric thought, and should be utilized at this point.

A number of separate trends in the treatment of alcoholism are now being followed by psychiatrists and specialists in internal medicine who are interested in the application of psychology in therapy with particular reference to personality factors and the "mind-body" problem. Patients with neurotic personalities and immature emotional development are the ones who respond most favorably to psychological treatment. Those patients with psychopathic personalities who may be pathological liars should not be included in the group considered suitable for psychotherapy, nor should one include patients suspected of being psychotic in the clinical sense. Psychotherapy has not been very successful in the treatment of schizoid individuals who are also alcoholic, or patients in the manic-depressive group. Mental defectives and cases of constitutional psychopathic inferiority are not promising subjects for psychotherapeutics. An unhappy childhood, especially where one or both of the parents have been alcoholic, may predispose the individual to the behavior pattern of drinking. The social and occupational history, the patient's attitude toward himself and his environment, are the best criteria of selection for treatment. For patients whose history suggests deep-rooted, unconscious difficulties, psychoanalytic treatment may be more suitable. These cases may be differentiated from cases eligible for less intensive psychotherapy. The intelligence and cooperation of the patient require careful consideration before any psychotherapy is recommended.

One great present need is additional hospital space where alcoholics can be treated, especially those beyond the acute stage. The attitude of almost all general hospitals is the same as that of the Boston City Hospital; they are grudgingly accepted (which is natural) and are discharged as soon as it is decently possible. In Boston there is one institution which cares for the alcoholic patient, the Washingtonian Hospital. This institution was organized on November 5, 1857, and incor-

porated on March 26, 1859. In 1940 it was reorganized with a consulting and visiting staff of prominent specialists. It is the sole institution which cares for alcoholic patients willingly, meanwhile maintaining its charges at a level which the average non-pauper alcoholic can pay. Perhaps the greatest value of the Washingtonian Hospital is that it will continue to attempt to rehabilitate patients who are gainfully employed and who are valuable to their employers. Except for this hospital, there is no other non-municipal hospital in Greater Boston for the care of alcoholics except several psychiatric institutions, where the expense is naturally greater.

The general attitude toward a chronic alcoholic must embody the concept that his condition is tantamount to a mental illness and that his personality has been damaged at some time in some way. The pattern of drinking belongs in the realm of symptomatology. One of the chief errors in the past has been to consider the problem of drinking as an entity without consideration of the patient's total personality in its situation. His thoughts, emotions and reactions must form the material of therapy. The drinking as behavior may then be evaluated in its true significance and usually as a symptom. In any science, a method is valid only in so far as it is appropriate for use with the material under examination. A single chemical analysis cannot be applied for all substances. Likewise, one particular method of treatment may be most applicable to one particular type of alcoholic patient. Much important clinical and psychiatric investigation remains to be carried out along these lines in order to answer many questions that arise in this connection. Preventive measures can be easily formulated, but they are difficult to put into practice. A rational program of mental hygiene in childhood along with the warmth of affection and the satisfactions and security of normal family life are the best safeguards against the development of alcoholism. The facing of and adjustment to reality, the building of character and the maturing of a normal personality are more likely to be accomplished under such conditions.

The prognosis in chronic alcoholism is not very favorable. It is not possible to cure alcoholic addiction easily, but with understanding and cooperation on the part of physician, patient and friends, it is often possible to lengthen the intervals between drinking episodes until they occur only at widely separated intervals. The need for an escape pattern of living can be lessened and when the obsessional features of drinking are understood it is often possible for the patient to make a moderately effective social readjustment.

Parents, husbands, wives, brothers, and sisters find it hard to believe that incorrigible drinkers are not perverse and deliberate. It is difficult to believe that their unfortunate loved ones are relatively powerless to resist the urge to drink. They must realize that the sufferer has a defective ego structure similar to that of a person who unconsciously seeks escape in drug addiction, in some forms of insanity or, more permanently, in suicide. The personality defect is not necessarily incurable, but at best its treatment may require months or even years of patient, loving sympathy and support. The aim of treatment is to repair the damaged personality and not by any means to punish the alcoholic. Only when this is realized by patient and relatives is a cure possible.[19]

Summary and Conclusions

1) Alcoholism is a public health problem and one that is often related more or less directly to many other fields, such as sociology, economics and the law.

2) It is a symptom or result of other disturbance in the personality or the life situation of the patient, but it may also result in illness, with complications.

3) It often complicates or aggravates medical or surgical conditions with which it is associated and rarely improves them.

4) The actual incidence of alcoholism, its occurrence, distribution and importance as a complicating factor in other medical and surgical conditions, is unknown (though probably greater than one might suspect) due to the common practices such as: (a) the patient may conceal the amount and pattern of his drinking, or lie about it; (b) the physician may not be aware of it, or out of consideration or sentiment may conceal it or minimize it; and (c) friends and relatives tend to "cover up" and protect the alcoholic.

5) Although alcohol in itself may not be primarily toxic, it favors the development of other pathology in the body, especially

that associated with dietary deficiency or imbalance.

6) The commonest complications of acute alcoholism are surgical: contusions, abrasions, lacerations and fractures. Head injuries are extremely common. The commonest medical complications are gastro-intestinal disturbances, pneumonia, and conditions associated with dietary deficiency. Disorders of the nervous system are also common: polyneuritis, Wernicke's disease (and head injuries with resulting complications such as subdural hemorrhage, vascular accidents and convulsions).

7) The death rate in delirium tremens is extremely high. The incidence of alcoholic psychoses is substantial.

8) The general outline of alcoholism is becoming clearer and as it becomes more clear it is taking shape not so much as a disease, but as a syndrome or entity with definite limitations and relationships.

9) On account of its wide prevalence throughout the nation in all social levels, alcoholism is now coming to be regarded as a major public health problem along with syphilis, tuberculosis and cancer. It is as difficult for the doctor to treat and as dangerous to the patient as a psychosis, yet alcoholics are not legally insane.

10) The prognosis has always to be conservative. Many alcoholics do not realize anything is wrong and do not feel the need of treatment, or do not seek it, or accept it, and, if it is offered them or if it is attempted, they do it because someone else (a member of the family or an employer) insists, and are often uncooperative or actually resistant to treatment.

11) The best medical treatment consists of rest, elimination, restoration of fluid, and acid-base balance (many alcoholics are in acidosis), and the use of sedative and analgesic drugs. For the chronic alcoholics vitamin therapy and psychotherapy are indicated. In certain cases institutionalization is necessary.

12) Since alcoholism is so prevalent, so far reaching a personal and public health problem, it is obviously a matter of concern because of its role in shortening life and causing death (and accidents).

13) It might be worthwhile to consider the problem more thoroughly from a statistical or investigative point of view as a public health problem in association with persons or institutions especially interested in the problem, with a view to determining the total incidence of alcoholism, its effect on health and any relations that might be determined that would be helpful in preventing or lessening its ill effects, as one might do in the case of any other illness.

14) Public health agencies may very profitably give consideration to these possibilities as a means of raising and clarifying issues relative to alcoholism and medical care, and may well continue to supply leadership and support in maintaining and improving standards in the practice and principles of medical diagnosis and treatment. They may cooperate even further in innumerable ways with those who are interested in treating and studying alcoholism with the aim of understanding and preventing it.

15) Improved facilities for the treatment and study of alcoholic patients are needed very badly. There is at present almost nowhere one can send the alcoholic patient for diagnosis and treatment except to private institutions which are not economically possible for most people. Alcoholic patients are not welcome in most general hospitals, suitable treatment is often not available for them there, and they are not eligible to be committed in most states, hence state hospitals are often not suitable, as alcoholics resist classification with the psychotic or criminally insane population.

16) Further social, psychological, and medical-legal studies of alcoholism will be very worthwhile, especially if these are correlated with chemical and clinical studies.

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Further Studies in Dilantin Sodium Therapy of Epilepsy*

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Introduction

In January of 1940, one of us (J. L. F.)[1] published the preliminary observations of Dilantin Sodium therapy begun in a series of twenty-eight epileptic patients in May of 1938. This report corroborated the favorable results presented by Drs. Merritt and Putnam[2] in the first publication on this subject in September, 1938. However, attention was focused upon certain of the marked side reactions with this new medication. Similar results have been reported in some forty publications during the past two years.

After a period of almost three years' observation, it was deemed advisable to present a follow-up study which includes the continued observation of the old patients as well as additional patients now taking this medication.

The present paper, therefore, deals with the use of Dilantin Sodium in fifty patients, including both private and dispensary epileptics, who have been observed over a period of from six months to three years. Many of these patients had been attending the Out-Patient Epileptic Clinic at the Lakeside Hospital for years and a record of their attacks, previous therapy, and personality was carefully kept.[3]

The clinical experience with Dilantin will be presented from several angles: first, the method of administration and the dosage used, then the appraisal of the clinical worth of Dilantin as an anticonvulsant, and thirdly, a consideration of the side actions with some statement as to means of reducing these complications.

Method of Administration and Dosage

As is well known, Dilantin Sodium is commercially available in capsules, each 0.1 gm. (1½ gr.). At first this medication was pre-

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scribed in place of previous therapy (usually phenobarbital), the patient taking one capsule of Dilantin approximately three to four times daily. So numerous were the unpleasant side effects that patients sometimes discontinued the drug voluntarily. Experience has shown that the drug should be administered in gradually increasing amounts. It is our custom to add one capsule per day to the previous medication, at the outset. This is continued for a period of a week or two. If there is no reduction in the number of seizures, then an additional capsule is added, replacing one tablet of the previously used phenobarbital. This means that, during the second or third week, the patient is receiving two capsules of Dilantin, perhaps one after breakfast and the other after supper. This dosage is maintained during the fourth and fifth weeks. If the seizures have been reduced, no additional Dilantin is used. If the number of attacks has not been lessened, then a third capsule of Dilantin is prescribed. Thus, at the end of one month or six weeks, the epileptic patient is receiving one capsule of Dilantin t.i.d., at which point his previous medication has been greatly reduced or discontinued. If necessary, a fourth or a fifth capsule of Dilantin is prescribed.

With this graduated program of administration, most patients tolerate Dilantin satisfactorily. One may thus compare this acquired tolerance for Dilantin with the manner of learning to smoke a cigar. If, for the first time, a person smokes a whole cigar, he is very apt to become nauseated, weak, and uncomfortable. However, if he takes a few puffs first and increases the time interval of smoking gradually, he may in a period of time gain great enjoyment from cigar smoking. As one looks back upon our experience over a period of three years, it is remarkable that so few patients complain about Dilantin when it is administered gradually, as contrasted with those who protested when it was prescribed in larger doses at one time.

It is to be noted that, as the Dilantin is increased, the previous medication is decreased. No abrupt changes should be made. In patients who do not respond to Dilantin alone, the addition of phenobarbital has proved valuable in some instances. This synergism of phenobarbital and Dilantin has been favorably reported by Cohen, Showstack, and Myerson,[4] by Robinson and Osgood,[5] by Pratt,[6] and by Williams.[7] Two of our group of fifty patients have shown a more favorable response to this combination than to either drug alone.

The Clinical Influence of Dilantin Upon the Symptoms of Epilepsy

The details of the results in our fifty patients are summarized in the accompanying table. Our experience concurs with that of Merritt and Putnam[8] as reported in their second clinical paper. Briefly stated, eighteen of the fifty patients have shown a remarkable improvement. Four of them, previously suffering frequent attacks, have shown a complete freedom from seizures. Fifteen patients have responded with slight or moderate improvement; sixteen have shown no appreciable change, and one may be classified as worse.

Let us consider the clinical results from the criteria of the specific symptoms of the disease:

Major Attacks—In patients in whom grand mal seizures predominate, the results have been satisfactory. The excellent benefit derived by certain individuals is illustrated by the case history of Miss H. P. itemized in the previous paper. At the time of the initial writing, this young woman had been free of attacks for one year. At the present date, we may state that she has been free of convulsions for almost three years.

Minor Attacks—Along with the experience of others, we find that the petit mal reactions are not so readily removed as are grand mal attacks. Those who have enjoyed a freedom from grand mal are likely to have a marked reduction in the petit mal attacks also. However, patients whose illness is characterized by numerous petit mal seizures have shown less improvement.

Psychic Equivalents—One can make no positive statements about the influence upon this symptom of epilepsy. The average epileptic patient has an assortment of attacks: grand mal, petit mal, and psychic equivalents. If Dilantin has reduced the grand and petit mal attacks, it has a similar value for the psychic states.

Personality—An outstanding feature of the benefit of Dilantin is the amazing improvement of the personality of patients favorably affected. Whereas certain epileptic patients who have shown a reduction of seizures

through bromide or phenobarbital medication may develop a dullness and decreased mental efficiency, the reverse is true of the Dilantin-treated cases. In several striking instances, individuals who had been dull, fearful, and disinterested in outside activities, have emerged as bright, confident, and capable individuals.

Case Abstracts

H. P. had previously been slow, stayed close to her bed, and her chief concern was recovering from one attack and anticipating the next. Today, this young woman is a neatly dressed, happy, and efficient personality, contributing to the welfare of her family.

J. A., a former inmate of an epileptic colony, who first appeared walking slowly, with an acne-marked face and dull expression, is now a keen, ambitious, and happy employed individual. He has been free of both major and minor attacks since Dilantin was started a year ago.

This improvement in personality may be ascribed to various factors: the withdrawal of previously used sedative drugs, the removal of the fear of attacks with release of ambition and development of outside interests, and possibly the awakening effect of Dilantin as compared with the lethargic influence of other medication. The improvement in personality with the use of Dilantin has been reported by Ross and Jackson[9] upon a group of institutionalized epileptics. These authors mention not only a reduction in seizures but an improvement in conduct. They state that there was no significant influence on intelligence ratings. We had previously reported that psychometric tests taken after Dilantin showed no unfavorable change compared with previous tests of the same patients taken by our psychologist, Mrs. Barnes.[10]

Side Actions of Dilantin Sodium

The many side actions which patients may show may be grouped under various headings as follows:

Central Nervous System—In our first paper, we stressed the many nervous manifestations of patients who were receiving Dilantin. A large percentage complained of an inner feeling of nervousness and exhibited some outward sign such as tremor. Some individuals spoke of hypersensitiveness even to the point of wakefulness. Several exhibited personality changes with irritability and suspiciousness which amounted, in one or two instances, to a definite paranoid reaction.

The further experience permits us to state that the majority of these symptoms are transitory. No patients have shown any prolonged personality disorders. Indeed, through the graduated administration of the drug, the more severe symptoms have been greatly minimized.

The majority of patients still show moderate nervous reactions. One has had a temporary diplopia. Tremor of the extended hands is common even with the "step-up" administration of the drug. The nervous symptoms are present during the first month and may require a reduction in dosage.

Gastro-Intestinal System—One of the commonest side actions of Dilantin is a swelling of the gums, first described by Kimball,[11] and discussed by Frankel[12] and by Merritt and Putnam.[13] This swelling is in the nature of a hyperplasia which ranges from a slight puffiness to a marked redundancy and, when present, usually affects the anterior aspect of the lower gums. Such puffiness attains a certain degree and remains stationary over a period of time. We have not observed a progressive enlargement of the gums as Dilantin was continued over a period of years. Though there is a superficial resemblance of this gum swelling to the pathologic gum changes of scurvy, yet studies by Merritt and Foster[14] and the researches of the Parke-Davis Laboratories[15] have shown that there is no relation between such gingival hyperplasia and the plasma Vitamin C content.

A few patients have developed gastro-intestinal complaints. Several have complained of disagreeable sensations in the mouth and some are nauseated. An occasional symptom is a distinct loss in weight. This is often accompanied by anorexia and by gastric distress. Such weight loss may reach five to ten pounds in a month or two even in thin individuals. This "emaciation" may occur though the patient has been distinctly benefitted by the anticonvulsant properties of the drug. For example, E. C., a slender adult of thirty-five, lost ten pounds, from 121 to 111, in a period of three months despite the freedom from seizures during that period. There was some loss of appetite, but her major com-

plaint dealt with nervousness: "I am unstable, jumpy; my hand trembles so that I cannot take food". Another patient lost about ten pounds in several months. Her chief complaints were: "I have a bad taste in my mouth as though there were flies in it; I have heartburn". These symptoms were so marked that the drug had to be gradually discontinued. In the case of E. C., Dilantin was prescribed again after a period of months and has been tolerated better. Merritt and Putnam[13] advise the use of dilute hydrochloric acid to reduce symptoms of indigestion.

Skin—As mentioned by other writers, there may be a transitory rash at the outset. We have seen this rather rarely. In one instance, there was a rash with an eosinophilia of seven per cent twelve days after Dilantin was started. After the drug was omitted for a short period, the rash disappeared. Readministration caused no further skin manifestations.

Hematopoietic System—There may be eosinophilia accompanying the dermatitis. In one patient we noted also a relative neutropenia and lmphocytosis. With these exceptions, we have seen no clinical or laboratory evidence of blood dyscrasia from Dilantin.

Miscellaneous

Although not directly concerned with the use of Dilantin, we may take this occasion to mention the creation of a sheltered workshop for epileptics. Those who treat epileptic patients realize the need for psychotherapy and occupational therapy as a supplement to the use of drugs. To meet this need, there has been established in Cleveland a therapeutic work program for epileptics called the Auracraft Shop. Epileptic patients previously idle and unemployable have been given the opportunity of training, social contact, and the joy of work plus small monetary compensation. Coupled with the gains from Dilantin medication, the epileptic patients have shown a favorable response to the constructive work program.

Comment and Conclusion

The present paper represents the results of further observation of Dilantin Sodium therapy of epilepsy. These additional studies substantiate the earlier opinion of the favorable results of this medication. As an anticonvulsant, its clinical value is distinctly superior to previous medication. Not only patients with idiopathic epilepsy, but also those with the organic form of the disease have derived benefit. Our estimate agrees with the recent appraisal of Dr. Lennox,[16] who considers Dilantin Sodium as an outstanding medication for epilepsy.

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Name & Age	Type and Duration	Pre-Treatment Average Frequency Per Month G. M.	P. M.	Results of Dilantin: Change in No. of Attacks	Personality	Complications
H. P. 30	Idio 15 yrs.	4	Many	Complete cessation.	Remarkably improved.	None.
J. A. 24	Idio 4 yrs.	6	Several	Complete freedom.	Phenomenal change.	None.
M. A. 20	Idio (?) 4 yrs.	1	Occasional	Improvement.	Regained confidence.	None.
B. T. 16	Idio 5 yrs.	Many		One attack in two years.	Remarkably improved.	Moderate weight loss, then slight increase: 123-110-114. Moderate gum swelling.
R. B. 34	Idio 4 yrs. (Questionable traumatic)	1	Several	Complete freedom.	Mentally alert.	None.
J. K. 28	Idio 6 yrs.	Many		Moderate improvement with phenobarbital; Dilantin added—attack free until medication discontinued.		Weight loss from 153 to 145. Some wakefulness at first. No other complication.
A. C. 44	Idio 13 yrs.	1	Several	Complete cessation (Treated since Oct., 1940).	Improved.	None.
L. F. 10	Idio 5 yrs.	Occ.	Frequent	Moderate improvement with phenobarbital. Dilatin began in Sept., 1940—Has been attack free.		Slight gum swelling.
E. C. 43	Idio 2 yrs.	3		Slight improvement with phenobarbital. Dilatin resulted in definite reduction of attacks. Discontinued because of side actions; attacks recurred. Dilatin resumed with reduction of attacks and fewer side actions.	Nervousness, irritability with Dilantin. But, when discontinued and later resumed, these side actions were less marked.	Weight loss (121-111), nervousness and irritability, moderate tremor of extended hands.
C. C. 20	Idio 6 yrs.	1-4		Little improvement with phenobarbital. With Dilantin, one attack in six months.	Definite improvement.	Moderate gum swelling.
M. L. 54	Idio 34 yrs.	Many	Many	Considerable improvement.		None.
H. B. 24	Idio 8 yrs.	Numerous	Many	Moderate reduction of seizures, combining phenobarbital and Dilatin.	Improved; more interested in responsibilities.	Slight gum swelling.
J. G. 16	Idio 5 yrs.	1	Occasional	Moderate improvement.		Weight loss from 116 to 108; then regained to 116.
E. M. A. 17	Idio 11 yrs.	Frequent	Frequent	Moderate improvement.		Moderate gum swelling. Weight loss—striking at first (112-104); then weight upward to 109.
T. M. 51	Idio 16 yrs.	Infrequent		Slight improvement.		Anorexia (temporary). Recently a rash appeared.

Name & Age	Type and Duration	Pre-Treatment Average Frequency Per Month G. M.	P. M.	Results of Dilantin: Change in No. of Attacks	Personality	Complications
L. B. 25	Idio 14 yrs.	1	Occasional	Moderate improvement with Dilantin at first. Continued use showed less sustained improvement. Dilantin and phenobarbital combined were no more efficacious than either alone.		Moderate weight loss (152-145). No other side actions.
C. F. 31	Idio Since infancy		Frequent	Moderate improvement with phenobarbital. Dilantin begun Nov., 1939. Striking improvement at first, then return of seizures with only slight improvement.		Weight loss from 135 to 130, then increase to 137½.
R. W. 33	Idio 8 yrs.	Many	Many	Slight improvement.	Patient more alert and more active since taking Dilantin.	Moderate gum swelling.
G. O. 35	Idio 20 yrs.		Many	Slight improvement with Dilantin at first, but this was not continued. Attacks remain unchanged.	There is a serious personality change, which antedated Dilantin.	Subjective discomforts, but no objective complications.
F. H. 33	Organic 7 yrs.	Occ.	Occ.	Little or no change with Dilantin.		None.
To. Mu. 40	Idio 30 yrs.	Occ.	Occ.	Unimproved.	Peculiar mental reaction, probably inherent.	
A. G. 19	Idio 5 yrs.	3-4		Period of study too brief for results to be conclusive.		
E. Z. 20	Idio 9 yrs.	8-12	10-20 per day	Period of study too brief for results to be conclusive.		
A. L. 37	Idio 19 yrs.	At first, infrequent seizures. Frequency increased.		Period of study too brief for results to be conclusive.		
S. S. 34	Idio 22 yrs.	3-6	Rare	1 attack in 2½ yrs. 5-6 "sensations" per month. Marked improvement.		Dizziness and ataxia at first.
M. S. 33	Idio 20 yrs.	1-6	1-6	Marked improvement: 4 attacks in 2½ yrs. Premenstrual "jerks".		Irritability at first.
B. V. 24	Idio 17 yrs.	7-8		Marked improvement; no attacks for 3 months.		Redundant gums.
H. H. 22	Idio 18 yrs.	10	30	Marked improvement.		Dizziness, palpitation, and tremor at first.
S. B. 40	Idio 33 yrs.	1-30	1-60	Marked improvement: Attacks reduced to 6 the first year, then further reduction.		Shakiness and ataxia at first.

Name & Age	Type and Duration	Pre-Treatment Average Frequency Per Month G. M.	P. M.	Results of Dilantin Change in No. of Attacks	Personality	Complications
S.N. 29	Idio 21 yrs.	23	Many	Marked improvement: Attacks reduced to 4 the first year.		None.
J.C. 26	Trauma Bullet 13 yrs.	2		6 attacks in a year and a half—marked improvement.		None.
T.T. 22	Idio 6 yrs.	7		Marked improvement: No attacks after 4 months on Dilantin.		Transient sore, hard palate, occasional diplopia.
L.M. 22	Idio 19 yrs.	8-30		Marked improvement: Only 1 attack since taking Dilantin.		None.
J.D. 21	Idio 20 yrs.	1 in 3 mos.	9	Marked improvement: No attacks for 3 months.		None.
J.V. 34	Idio 34 yrs.	8-12	Many	Moderate improvement: No grand mal attacks; about 10 petit mal per month.		Redundant gums.
F.K. 31	Idio 6 yrs.	1		Number about the same, but less severe.		None.
L.K. 21	Idio 5 yrs.	1 in 2 mos.	Many	Moderate improvement: No grand mal attacks; 2-4 petit mal per month.		None.
Ri. Bl. 26	Idio 12 yrs.	0-4	2-8	Same number of grand mal, but no petit mal.		Dizziness, absent-mindedness. Dilantin discontinued.
P.D. 31	Idio 20 yrs.	Irregular	8-12	Slight improvement in general status. Same number of attacks.	More alert.	None.
Ho. Be. 32	Questionable trauma 3 yrs.	3-8	4	Attacks about the same.		Transient maculo-papular.
So. Bo. 31	Idio 23 yrs.	0-16	0-7	Attacks about the same.		Acne, weakness, irritability at first.
La. Ba. 32	Idio 13 yrs.	1-12	1-6	Attacks about the same.		Sore mouth and gums for two weeks.
A.R. 17	Idio 11 yrs.	0-10	0-5	Attacks about the same.		None.
D.B. 29	Idio 17 yrs.	1-6		About the same.		None.
D.G. 14	Idio 7 yrs.	1-60 per day		About the same.		None.
M.B. 27	Idio 14 yrs.	1 every 2 mos.		Slightly worse; 2 attacks per month.		Shaky at first; Questionable hyperplasia of gums.
D.S. 17	Idio The past yr. and also from age of 1 to 7	1 every 2 mos.	3-4	About the same.		None.
A.T. 34	Idio 11 yrs.	0-2	2-3 per day	Same or slightly worse.		Nervousness.
Ma. Bo. 40	Idio 27 yrs.	4		About the same.		Macular rash 12 days after Dilantin started. Eo-7 simul.
M.F. 46	Idio 20 yrs.	1	2-3	Worse: 3 times as many grand mal for 1 yr.		Decreased memory, tremor, weakness, dizziness, loss of pep.

Pathologic Lesions of the Brain Associated With Chronic Rheumatic Endocarditis and Accompanied by Psychosis

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In 1932, Winkelman and Eckel[1] described pathologic cerebral lesions associated with rheumatic fever and with neurologic and psychiatric symptoms. The changes consisted mainly of proliferative endarteritis of the smaller cortical vessels with cellular atrophy of the surrounding tissue, and with gliosis. In 1936, Breutsch[2] and in 1939, Breutsch and Bahr[3] described similar lesions in the brains of individuals with chronic mental illness. These were accompanied by chronic rheumatic endocarditis and in some cases by vascular lesions in other organs. Obliterating endarteritis of both small and large cerebral vessels appeared to result in gross and microscopic infarcts; the latter were numerous and widely distributed and resulted commonly in gliosis. The psychosis took a diversity of forms. The diagnosis made most frequently was dementia praecox. These observations were confirmed essentially by Kernohan, Woltman and Barnes[4], and by others.

During the year 1940, in the course of 161 post-mortem examinations at the Western State Hospital, rheumatic endocarditis was recognized grossly and confirmed microscopically in five cases. There was one instance of spontaneous thrombosis of the abdominal aorta and mesenteric arteries in the absence of marked atherosclerosis. Infarcts of lungs and kidneys were the rule; one kidney showed focal occlusion of glomerular loops like that seen in disseminated lupus erythematosus. In no case was there evidence of bacterial endocarditis.

In every instance, pathologic changes of varying degree were found in the brain. In two cases, gross infarction occurred in the region supplied by the middle cerebral artery. In one of these cases, occlusion appeared to have resulted from embolism; in the other, trauma in a person with severe rheumatic endocarditis resulted in infarction, apparently on a basis of thrombosis. Diffuse changes, which varied in degree with each case, consisted of proliferative thickening and occlusion of small cortical vessels, with miliary infarction and, occasionally, gliosis. In one instance, perivascular proliferation of lymphoid cells was striking. In no case was there an appreciable degree of cerebral atherosclerosis.

The psychiatric diagnoses made were: psychosis with cerebral arteriosclerosis; manic-depressive psychosis in 2 cases (in one case there were many catatonic features); psychosis with cerebral trauma; psychosis with convulsive disorder (convulsions began at the age of 40, 2 years before admission to the hospital; a rheumatic basis of the seizures is not dogmatically insisted upon, but is conceivable).

The age at which psychosis appeared varied from 25 to 45 with an average of 34.

The apparent psychogenic importance of rheumatic vascular disease varied with each case. The occurrence of trauma in one instance and of convulsive disorder in another has been mentioned. In another case, in a person known to have been of unstable personality, psychosis appeared in severe form, subsequent to the onset of the rheumatic process.

There is no question that pathologic lesions occur in the brains of some persons afflicted with rheumatic cardiovascular disease. The difficulty lies in making the diagnosis of rheumatic endocarditis, and in predicting, on the basis of the cardiac lesion, the nature and degree of cerebral vascular involvement. A comparison may be drawn to cerebral arteriosclerosis, in which an attempt is made to estimate the nature and degree of cerebral vascular change on the basis of neurologic examination, inspection of the retinal vessels, palpation of the peripheral arteries, and so

forth. A further difficulty arises in the diversity of psychiatric diagnoses made in the presence of rheumatic endocarditis; at present there appears to be no psychiatric syndrome characteristic of rheumatic vascular disease. It is, of course, possible that such a syndrome may some day be recognized. This situation was dealt with by Kernohan, Woltman and Barnes,[4] who stated: "That a psychosis may be intimately related to endocarditis and to the ensuing cerebral damage will be granted readily, even if such a psychosis cannot be recognized solely by its clinical features. After all, the pattern of a psychosis is determined in a large measure by the possible reactions of the personality."

There may be justification for considering a diagnosis of psychosis with rheumatic vascular disease in cases of psychosis in which rheumatic endocarditis can be detected and in which no other significant organic lesion is found. In so doing, one would of course run the risk of including in the aforementioned classification cases of functional psychosis which occurred coincidental with non-psychogenic rheumatic vascular disease. It should be recognized that the same is true of cerebral arteriosclerosis, in which condition the psychosis may not have a constant relation to the degree of vascular change, but may depend more closely on the "possible reactions of the personality."

Case Reports

The significant data of two illustrative cases are summarized briefly:

Case 1—No history of rheumatic fever was elicited. Between the ages of 25 and 30, a period of depression occurred; a second took place between the ages of 35 and 40. The final illness appeared at the age of 43 and was characterized by irritability, peculiar mannerisms, delusions of persecution, auditory hallucinations, and defective insight. The patient appeared preoccupied and distant, and muttered to herself. The psychiatric diagnosis was: manic-depressive psychosis, with many symptoms suggestive of catatonic dementia praecox.

Physical examination on admission to Western State Hospital, 25 years following the onset of the final illness, at the age of 68 revealed moderate enlargement of the heart, with a blowing sytsolic murmur of the mitral area and with auricular fibrillation. Death occurred, a few months following admission, from cardiac decompensation and was preceded by persistent tympanites.

Post-mortem examination revealed mitral rheumatic endocarditis of severe degree, with vegetations. There were infarcts of the lungs and kidneys, and there was thrombosis, without marked arteriosclerosis, of the abdominal aorta and mesenteric arteries. The bowel was discolored and distended. The brain, with its vessels, was not remarkable grossly.

Histologic examination showed rheumatic lesions of the valves. There were a few histiocytes in the myocardium and in the intima of the aorta beneath the point of attachment of the thrombus. The brain showed widespread proliferative thickening of the small cortical vessels. An occasional perivascular collection of lymphoid cells also was seen. There was patchy atrophy of the cortical cells. Occasionally, a senile plaque and a cortical cell containing lipochrome were seen, to a degree expected at the age of the patient. The sciatic nerves contained a few miliary infarcts.

Case 2—The history is of questionable reliability. At the age of 8, the patient was said to have had a paralysis of the left side of his face; he was said to have had convulsions as a child. While serving a jail term for drunkenness, he was observed standing in his cell mumbling and staring into space, and he could not remember which was his bunk. He was admitted to Western State Hospital a few days later, at the age of 50. He answered most questions by mumbling to himself and gesticulating.

Examination revealed enlargement of the heart and irregularity of the rhythm. There were left hemihypesthesia and hemiparesis. The patient suffered repeatedly from left hemiplegia (the hemiplegias occurring in rapid succession) with loss of consciousness, with partial recovery from paralysis between attacks; he expired during one of them one month following admission. The psychiatric diagnosis was: psychosis with cerebral arteriosclerosis, with thrombosis.

Post-mortem examination showed severe mitral and mild aortic rheumatic endocarditis. The lungs and kidneys contained infarcts. There was softening of the region supplied by the right middle cerebral artery.

Microscopic examination revealed rheumatic valvulitis. In the kidneys there was focal hyaline occlusion of loops of the renal glomerular tufts. In the region of gross cerebral infarction there were: necrosis, some hemorrhage, and proliferation of astrocytes and microglia. There was proliferative thickening of small vessels throughout the brain; this was most marked near and in the area of gross infarction. Perivascular proliferation of lymphoid cells was striking.

There was moderate, diffuse pyknosis of the cortical nerve cells.

Summary and Conclusions

In some persons having rheumatic endocarditis, cerebral vascular lesions, focal and diffuse, occur. These consist of proliferative, thrombotic and embolic occlusions of blood vessels of all sizes, with destruction of the tissue which the vessels supply. These changes are found, in varying degree, regularly in persons with psychosis in whom, at post-mortem, rheumatic endocarditis is recognized.

The apparent psychogenic role of rheumatic vascular disease varies greatly with each case. The problem arises of considering a diagnosis of psychosis with rheumatic vascular disease in persons having psychosis coincidental with rheumatic endocarditis. Making such a diagnosis offers possibilities, but involves certain limitations, which must be borne in mind.

Addendum

Since completion of this paper, post-mortem examination has been performed in a case which perhaps should be classed as Libman-Sachs' disease, or non-bacterial endocarditis, rather than rheumatic. The patient, a white man aged 58, had complained of convulsive disorder since the age of ten. For several years he had been confused, forgetful, and irritable, and had been disposed to attack others following seizures. There was no history of rheumatic fever or chorea. Forty days before death, thrombosis of the superficial veins of the right thigh, without pain, but with edema of the extremity, occurred. The only other symptoms were weakness and a moderate degree of fever. No further observation of note was made until the patient died rather suddenly. Post-mortem examination revealed scarcely any general arteriosclerosis. The mitral valve showed no appreciable deformity; however, along the line of closure were pink pearly vegetations. Microscopically, these consisted of vascular connective tissue, but contained no bacteria.

The aforementioned thigh veins contained partially organized thrombi, but no inflammatory reaction was seen. In the brain was a recent, non-hemorrhagic softening of the superficial portion of the right temporo-occipital region. This had occurred in the absence of an appreciable degree of cerebral arteriosclerosis. Microscopic examination revealed, in addition to the aforementioned infarct, disseminated, minute softenings about small vessels; these appeared to have been occluded as a result of a proliferative process. Some of these lesions were old. About them, varying degrees of injury of nerve cells and of gliosis were seen.

This case is not considered a typical case of acute disseminated lupus erythematosus. It is, however, not characteristically rheumatic, and it deserves the diagnosis non-bacterial endocarditis with general and cerebral vasculitis.

The relation of cerebral lesions in this case to symptoms is, of course, subject to debate. At least it seems worth while to examine the brain as well as the heart, kidneys, and other organs, in which lesions have commonly been found, in "general vasculitis" of a type not characteristically rheumatic, and associated with non-bacterial endocarditis.

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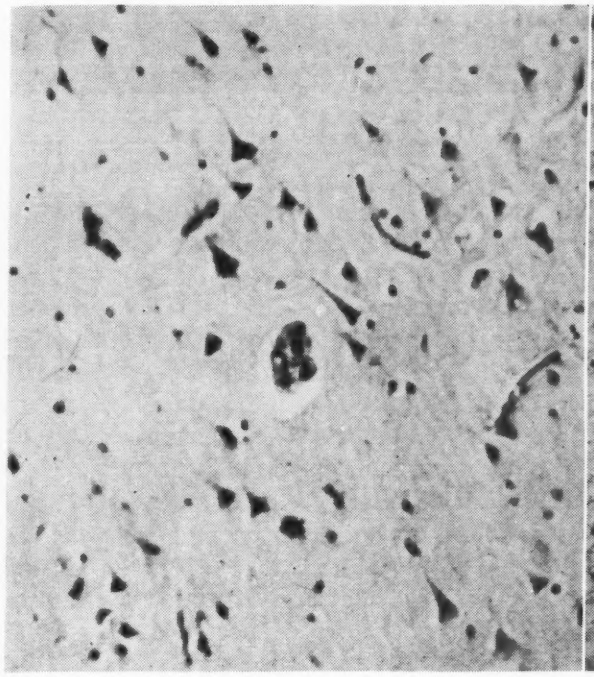

Figure 1. Proliferative thickening of small cortical vessel. H & E, x 300.

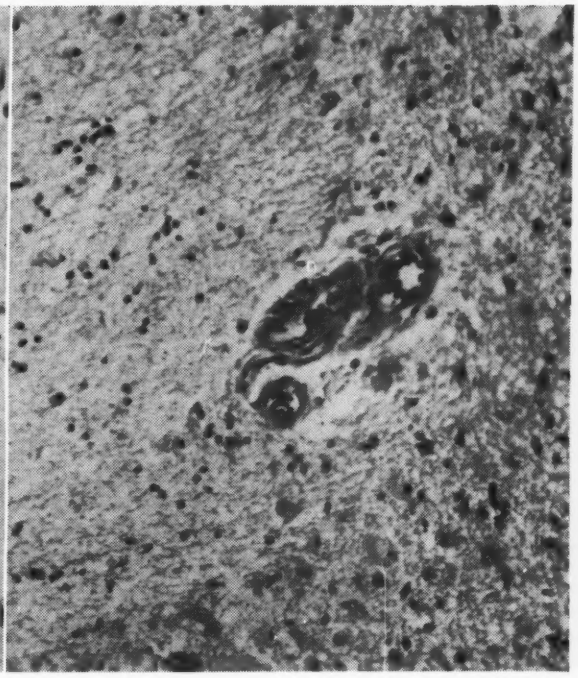

Figure 2. The same; infarction and glial reaction. H & E, x 200.

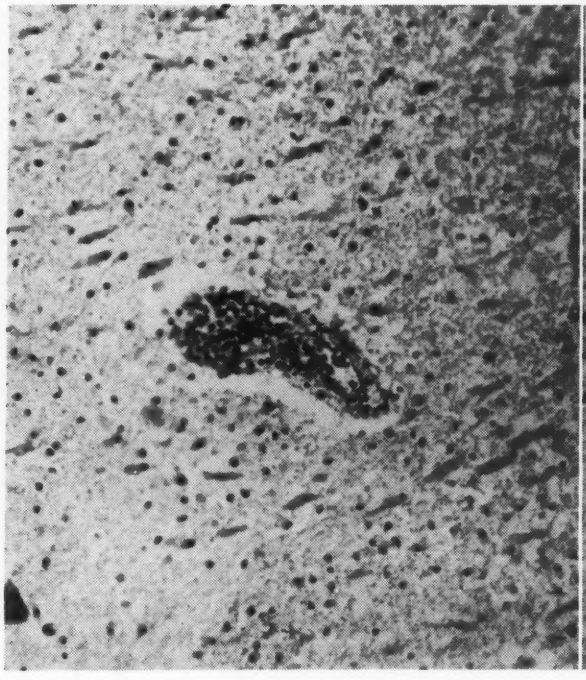

Figure 3. The same; infarction, glial reaction, and perivascular lymphocytosis. H & E, x 200.

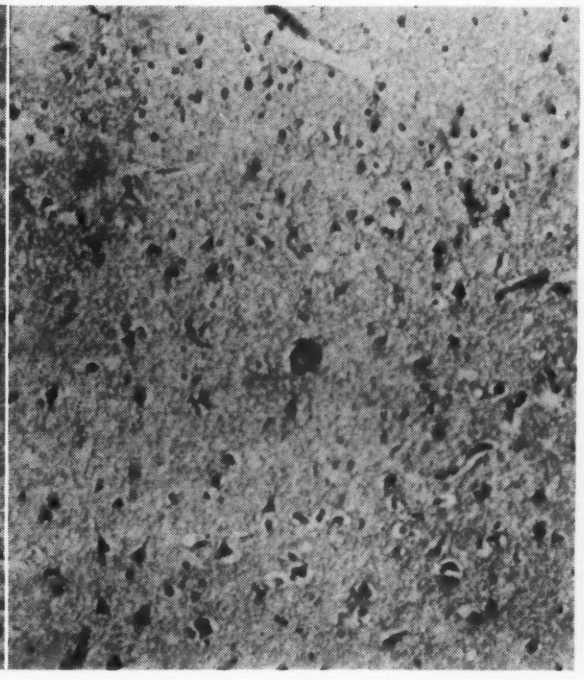

Figure 4. Thickening of wall of small cortical vessel; atrophy of surrounding zone of tissue; early gliosis. H & E, x 200.

Diagnosis and Treatment of Neurosurgical Lesions of the Spine, Spinal Cord and Cauda Equina

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An essential prerequisite to a skillfully performed neurosurgical operation is an accurate diagnosis of the disorder to be mechanically corrected. The diagnostic responsibility weighs heavily on anyone who presumes to cure by the scalpel. For this reason it is mandatory that he who assumes the burden of cure by surgery, must also assume the responsibility for an accurate preoperative evaluation of the problem at hand.

It has been well said that the neurosurgeon is more of a general surgeon than the general surgeon himself, so diversified are the anatomical locations he must be prepared to enter. In spinal cord, as well as brain surgery, the repairing mechanic (neurosurgeon) must be well qualified by training in anatomy, physiology, pathology, neurology, eye, ear, nose and throat diseases, to say nothing of general surgical principles.

The examination starts with a painstaking history. If thorough, this will give a definite clue as to the nature of the disorder. Weakness in the lower extremities, slowly progressive in nature, makes the examiner at once think of a lesion needing surgical care. Pain, as a symptom of neurosurgical disease of the spine, is not so common, nor is it liable to be so widely distributed. More often pain is localized to the precise segment of the lesion and to the side where it starts. If the lesion is in the thoracic or lumbar spine, the complaints will be directed to the legs, trunk, bowels and bladder. If in the cervical spine, then the disability extends to the arms.

A loss, or dimunition of sensation to pain, touch or temperature below the lesion, with a sharp line between the normal and abnormal segments may accurately localize a spinal cord lesion.

Lesions above the tenth dorsal segment cause the phenomena associated with pressure on the long motor tracts—Babinski reflex, hyperactive tendon reflexes, ankle and patellar clonus. When the lesion is below this point, these findings are absent and the lower extremities show a flaccid paralysis.

The x-ray examination of the spine cannot be too much emphasized. In this connection I should like to say that x-ray examination of the skull in suspected disease of the brain is still far too often neglected. No operation should ever be undertaken on the spine without it first being thoroughly x-rayed.

Often it is necessary to use contrast media in the spinal subarachnoid space (iodized oil, thorium dioxide, or air) in the accurate diagnosis and localization of the lesion.

Iodized oils are the most widely and successfully used contrast media. Most animal and vegetable oils have the property of combining with iodine in very high percentages. Once combined they are very stable, only slightly irritating, and are relatively radiation opaque in proportion to the quantity of iodine in combination. Generally speaking, the iodized oil is injected into the lumbar spinal subarachnoid space between the third and fourth vertebra. Inasmuch as the oil is very heavy it can be moved to any desired location in the spinal canal by altering the position of the patient on the x-ray table. It is seldom necessary to inject this material into the cisterna magna and because of the potential hazard of a spinal puncture in this location, it is to be avoided if possible.

Iodized oil is not a perfect material for use in the spinal canal because it is impossible to remove it without operating. It is never absorbed and there is evidence to make us believe that in some instances it sets up a chronic inflammation about the cauda equina.

Air may be injected as a contrast media

into the spinal canal in selected cases, but it is not very satisfactory. In thin, small patients it may give all the information desired. Thoratrast, colloidal thorium dioxide, in radiation active and irritating, and too dangerous to use in quantities sufficient for the diagnosis of spinal disease.

Examination of the spinal fluid is an essential part in the diagnostic routine of spinal disease. This should be performed with the same aseptic ritual as a major surgical operation. Meningitis and death are surely the penalty for a slovenly technique in carrying out this very important procedure. It is performed with the patient on his side and the neck free. A 19 gauge needle is used and the pressure accurately recorded with a water manometer. Following the measurement of the spinal fluid pressure the jugular veins are compressed. If there is no obstruction in the spinal canal between the point of puncture and the cranial cavity, the over filling of the veins in the cranial cavity will compress the cerebo-spinal fluid, forcing the fluid down the spinal canal, thereby raising the fluid pressure in the manometer. This is referred to as a negative Queckenstedt test and if an obstruction exists in the spinal cavity, there will be no rise in pressure in the manometer on jugular compression (positive Queckenstedt test). A massive displacement of the spine by fracture, or spinal cord tumors, give a positive Queckenstedt test by obstructing the spinal canal.

The routine laboratory examination of the spinal fluid should always include a cell count, quantitative protein estimation, test for syphilis and colloidal gold curve. It is unfair to the patient to perform spinal puncture without all of these examinations as a routine.

For the purpose of discussion, the common neurosurgical lesions of the spine, spinal cord and cauda equina may be conveniently divided into four large groups:

A. Congenital Malformations.
B. Neoplastic Disease.
C. Traumatic Lesions.
D. Inflammatory Masses.

Congenital Malformations

1) Embryologic protrusion defects may occur at any part of the spine, but are most common in the lumbar region. These protruding masses are always present at birth and are commonly referred to as meningoceles. Occasionally, they are composed of a sac of the meninges covered with skin and contain spinal fluid. More often the spinal cord, or cauda equina, is tightly adherent to the dome of the mass. It is then referred to as a myelomeningocele. As the fluid in the meningocele increases in quantity, its dome is lifted farther and farther from the spinal canal. This pulls the spinal cord and cauda equina from the spinal canal causing a paralysis of the legs, bladder and bowels.

Early operation, as soon as the patient is a good surgical risk, is advisable to correct the deformity and to save the cord and caudal structures from further injury.

The x-ray finding is that of an open posterior neural arch, as shown by absence of the laminae and spinous process of one or more vertebrae. The bony deformity is commonly referred to as a spina bifida, but not often are the spinous processes divided, more often the defect shows an absence of massive parts of the posterior arch. Simple spina bifida, as is often seen at the first sacral segment, is of no significance.

Congenital hydrocephalus quite commonly complicates a meningocele. So frequently are they associated that it seems very likely that protrusion defects from the skull and spine, such as meningoceles and encephaloceles, are caused by prebirth abnormal intracranial pressure.

2) Congenital tumors such as angiomas and lipomas may occur at any age, but are more apt to be noticed before adolescence. They give much the same symptoms and signs as neoplastic masses to be described later and are to be considered as surgical lesions.

Pilonidal cysts commonly occur over the sacrum and are the result of a hyperplasia of embryologic remains. They may be filled with epidermoid substances such as hair and epithelium, and are usually not connected with the dura and can be cured by complete extirpation.

Neoplastic Disease

1) Intramedullary tumors are those growths arising from the elements of the spinal cord itself. These are quite generally classified as gliomas and are usually infiltrating in nature. They frequently cause fluid

to form about them with associated cyst formation.

2) Extramedullary tumors grow from the structures about the spinal cord, that is, from the bone, nerves and meninges. The two most common tumors are the meningiomas which grow from the dura and arachnoid, or the neurinomas which grow from the sheath structures of the spinal nerves as they pass from the spinal cord.

Most extramedullary tumors are benign and can be completely and permanently cured by operation.

Occasionally, one finds a metastatic tumor pressing on the spinal cord which will give the same signs and symptoms as a benign tumor. Only the discovery of the primary malignant lesion, or a malignancy in the lung, will make one suspect the true nature of the growth.

The diagnosis of an extra or intramedullary lesion is made on the early symptoms of pain or tightness at the level of the tumor. If in the thoracic spine, the patient often complains of a feeling as though a tight belt were being drawn about the waist. Many times a spinal cord tumor will cause pain which may be attributed to sciatica, gall stones, appendicitis, kidney stones or hernia. In fact most every patient who comes to the neurosurgeon has had an operation for one of these disorders. Coughing, sneezing or straining aggravates the pain. Muscular weakness below the lesion is the next most common complaint. Later a loss of sensation to pain, temperature and touch can be demonstrated.

The spinal fluid examination often gives a positive Queckenstedt test. It will be high in total protein and may be discolored (xanthochromic).

Before operation the surgeon will practically always accurately locate the lesion by injections of 2 cc. of lipiodol, followed by fluoroscopy and x-ray films.

The operative procedure for spinal cord tumor is safe and one is usually rewarded by the most gratifying results. A patient completely paralyzed in both legs will walk as well as ever in a few months. No appreciable strength will be lost to the bony spine by the operation, as only the laminae and spinous process need be removed for gaining access to the tumor and these structures carry none of the weight of the body.

Traumatic Lesions

1) The neurosurgeon is only interested in injuries to the spine when the supporting structures press on the spinal cord, cauda equina or the spinal roots. The most common injury to the spine is the fracture which takes place through the body of the vertebrae. In this location, there are no large nerve elements and do damage is likely to take place to nerve structures unless the broken parts are displaced enough to press on the spinal cord or cauda equina. When this occurs, paralysis will be evident bilaterally below the the lesion.

Before the advent of surgeons trained in surgery of the spinal cord, it was common practice for the patient to be manipulated and placed in a plaster cast without the surgeon knowing whether he had aggravated the pressure on the cord or released it. It is now generally accepted as correct practice in all fractures with paralysis below the sixth cervical spine, to perform laminectomy so as to remove all compressing fragments before applying a plaster cast. Operation is to be performed at the earliest possible moment that the patient is considered a good operative risk. Only by this method of treatment can we assure the patient with paralysis from spinal fracture of the maximum recovery that is possible after cord injury.

Fractures of the spine above the sixth cervical segment with paralysis are best not operated on because of the terrific operative mortality, necessarily high because of a contused spinal cord in close proximity to the medulla oblongata. Blind extension of the neck with incorporation of the head, neck and thorax in a plaster cast is the treatment of choice. This treatment should be instituted as soon after the injury as is possible. It is entirely impossible to keep the broken neck parts from further traumatizing the spinal cord with the use of any other contrivance. Moderate extension by means of a canvas halter in addition to the cast may help. Weights which bruise the skin must be avoided, so it is usually best to keep them below three to five pounds, or even less if the skin will not tolerate this amount.

During the last five years much attention has been given to injury to the intervertebral disc as the cause for certain cases of sciatic

neuralgia. It is quite obvious that the reason this lesion was not discovered many years before, is that the broken disc cannot be demonstrated by the usual plain x-ray film. With the spinal injection of lipiodol we can now demonstrate fractures of the intervertebral disc where large fragments have become herniated from out of the disc space and encroach on the spinal canal. It is still impossible to see a fracture line through an intervertebral disc by any present known x-ray method, and simple fractures of this structure, without a herniated fragment, can be demonstrated only by open operation and direct inspection.

While a fractured intervertebral disc may often cause chronic backache and mild irritative phenomena about the cauda equina for years, yet the more common finding of unilateral sciatica neuralgia is not often evident until a fragment of the broken disc is extruded from the disc space proper and presses on a nerve root.

Microscopically, these fragments are very often composed largely of material from the very center of the disc, that part known as the nucleus pulposis. More often, however, the protruding fragment resembles other parts of the disc and occasionally small fragments of bone from the edge of the vertebral body are fastened to it. Then again, the protruding fragment most often has become degenerated into a piece of fibrinous material that is entirely unlike any part of a disc. In fact so seldom do these protruding masses resemble a normal nucleus pulposis, although occasionally they may, that they are now more accurately referred to as a herniated fragment of a fractured intervertebral disc.

While herniated fragments of intervertebral discs may occur at any level in the spinal column, they are most common in the lower lumbar and lower cervical regions. The great majority, at least 90 per cent, of these lesions occur in the lumbar region and three of every four in this region occur between the 4th and 5th lumbar vertebrae.

A herniated disc fragment in the lower cervical region will give pain in the arm and hand. In the lumbar spine it causes sciatic neuralgia inasmuch as the 4th and 5th lumbar roots go to make up the sciatic nerve. It appears as though there is a definite connection between this lesion and trauma as the pain often comes on suddenly after lifting a heavy object or falling in a sitting position. Usually, however, the initial distress is in the lower part of the back and it moves to the sciatic nerve distribution days, or even weeks, later. This distribution is down the back and lateral side of the thigh to the outer side of the calf and top of the foot and involves the small toes. It is a constant ache with sharp shooting stabs aggravated by moving, bending, coughing, or sneezing. Sometimes the patient can get up and about, but often is bedfast.

Objective signs are curvature of the spine, tenderness directly over the lower lumbar spine at point of injury and sciatic nerve tenderness. There may be atrophy of the muscles of the involved leg and a lost achilles reflex. Occasionally, in long standing cases, there is an area of diminished sensation over the outer side of the thigh and calf and the top of the foot and small toes. Once in a while there is a foot drop.

The spinal fluid examination often shows a moderately increased total protein. Injection into the lumbar spinal subarachnoid space of 5 cc. of lipiodol will show a filling defect in the spinal canal which indicates the location of the herniated fragment. This is best done by thoroughly observing the lipiodol movement beneath the fluoroscope.

The treatment of this disorder is by surgical removal of the herniated fragment. This can be accomplished without removing any of the structural weight bearing parts. The muscles are separated from the spinous processes and laminae of the vertebrae between which the lesion has been located. Small fragments of bone are removed from the laminae above and below the interspace but no part of the articular facets or spinous processes are disturbed. The spinal dura and the root compressed is lifted, and the protruding disc identified. Usually, there is a capsule over it which must be incised. This allows the fragment to be extracted with a forcep which leaves the root loose. The operation is concluded by suturing the muscles and skin with silk.

If the patient must return to hard labor, or if the disc seems to be badly damaged, in all likelihood he will be left with a structurally weak back. Then the operation is concluded by denuding the articular facets and

packing these joint spaces with bone chips, followed by placing massive bone grafts from the tibia between the laminae and spinous processes of the vertebrae involved. If a bone graft is necessary, then the patient is placed in a cast reaching from the ankles to the axillae in which he stays for ten weeks. If a bone graft is not done, the patient may get up in three or four weeks.

Inflammatory Masses

The common inflammatory diseases of pyogenic organisms, tuberculosis and syphilis, may produce abscess formation, or bone destruction in such a manner as to press on the spinal cord or cauda equina causing massive paralysis very similar to that caused by tumor growths. The signs and symptoms come on more quickly and are associated with the usual phenomena of inflammatory disease.

In the common pyogenic infection with abscess formation and cord pressure there is a fever, fast pulse and the other common signs of acute toxemia. While these abscesses are more often localized collections of pus outside the dura they may extend from anatomical locations outside the bony spinal canal, as for example, from a subdiaphragmatic abscess.

Tuberculous abscess of the spine is usually accompanied by tuberculous foci in the lungs or other parts of the body, and the x-ray will show destruction of the spine characteristic of the disease.

Syphilis is more apt to produce a massive bone deformity without pus formation. The spine is so weakened as to become displaced, permitting the spinal canal to be encroached upon. A positive test of the spinal fluid or blood for syphilis with the x-ray evidence, will inform the examiner as to the true nature of the disorder.

All of these lesions where paralysis exists must be treated by surgery. Laminectomy must be performed and the pyogenic abscesses opened. In a tuberculous abscess it is best not to open the abscess if the cord can be relieved of pressure without it. A cast should be applied and the general therapy used in treating this disease instituted. With syphilis of the spine wide laminectomy must be performed early, a cast applied and antisyphilitic therapy vigorously given. Treatment of the syphilis alone in the presence of paralysis will not produce a satisfactory cure. If operation is delayed too long, the compressed cord may be permanently injured.

Surgery on the spine, generally speaking, gives very gratifying results. Most of the lesions are benign and the operative shock and risk is negligible in well trained hands.

Regarding x-ray therapy of lesions of the spine, spinal cord and cauda, it may fairly be said that it is practically never curative, and at best serves only as a palliative, adjunct treatment to surgery. Its use should be recommended by the neurosurgeon after he has, by direct surgical inspection, determined the exact nature of the lesion to be treated.

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Book Reviews

PRINCIPLES OF ABNORMAL PSYCHOLOGY: THE DYNAMICS OF PSYCHIC ILLNESS. Maslow, A. H., and Mittelmann, B. New York and London: Harper, 1941. Pp. x 638. Price, $3.50.

Traditionally, a textbook of abnormal psychology is either a superficial descriptivistic account of various mental disorders or it is an academic presentation of disorders of memory, conation, cognition, affect, attention, perception and similar concepts.

This book, however, definitely breaks that tradition. It constitutes a presentation of experimental and clinical material adequately discussed to make clear the dynamics of mental abnormality and illness.

To orient the reader and to give a preliminary understanding of the need to comprehend the total personality, the book opens with a typical neurotic case history including symptoms, previous difficulties, childhood history, treatment and outcome, such as is encountered so frequently in the practice of medicine.

Discussion of this case is then given in concrete terms of the dynamics involved: of the conflicts, frustrations, disturbances of self evaluation and defense reactions manifested by the patient.

With this introduction the authors proceed

to discuss lucidly the fundamental concepts of abnormal psychology, the meaning of the term abnormal, what constitutes a normal personality and the various psychosomatic problems of motivation, emotion, etc.

Part II. is devoted to an elaboration of each of the various psychodynamic processes encountered in mental abnormalities and it also includes a discussion of experimentally produced behavior disturbances. Part III. deals with the etiology of psychopathology, covering genetic, constitutional, environmental, cultural and social factors, and the origin of psychopathology in childhood and in parent-child relations. Part IV. treats of the aims and the nature of psychotherapy, the technique employed and the use of suggestion, hypnosis and psychoanalytic therapy. The final section discusses the symptom syndromes, particularly the traumatic neuroses, phobias, obsessions, compulsions, disturbed states of consciousness, organic neuroses and the various psychiatric entities.

In addition to the lists of suggested readings appended to the individual chapters, there is an additional bibliography of 819 articles which were actually employed in the workup of this book. An excellent glossary is included and there are 2 appendices covering projective methods of examination and statistical data on mental disease. The book is well documented throughout with pertinent and informative case material and experimental work. It is interestingly written, easy to teach and, though it was written primarily for the college student, it is a textbook that the general practitioner and the psychiatrist can well afford to have ready at hand for frequent reference.

Milton H. Erickson, M.D. Eloise, Michigan

THE THERAPY OF THE NEUROSES AND PSYCHOSES: Socio-Psycho-Biologic Analysis and Resynthesis. Samuel Henry Kraines, M.D. Associate in Psychiatry University of Ill. College of Medicine; Assistant State Alienist, State of Ill. Diplomate of American Board of Psy. and Neu. Lea and Febiger, Philadelphia, 1941. Price. $5.50.

This book of 512 pages presents a new and thought-provoking method of subdividing the contents for discussion. The subject matter, as a whole, has been handled in an excellent fashion, while the author's style of writing is generally clear, readable, and usually so much to the point, that his descriptions of methods of therapy which he does not accept, are critically brisk and even curt.

Dr. Adolf Meyer in the Foreword aptly describes the book when he states, "Its sincere and practical spirit puts critical theory into the background without sacrificing the search for the realities and the opportunities for service. May the spirit of the reader join that of the writer." Certainly this book reveals the writer's spirit and enthusiasm in a manner which is not frequently encountered in the average textbook of psychiatry. Dr. Kraines mentions or discusses most of the conditions encountered by the practicing psychiatrist, and one feels that the author has achieved his purpose which is to prepare a reference textbook for psychiatry.

Since the psychoneuroses receive marked attention, and all forms of therapy are considered in detail, whether psychological, physiological or physical, this book should also be a valuable reference source to the general man in medicine. His phraseology and terminology is admirably suited for such purpose. Even the chapter on Psychopathology utilizes commonsense, everyday experiences to illustrate mental mechanisms.

From the standpoint of content, the first chapter deals with a general classification of psychiatric states, and while the author deplores classification per se, preferring to stress descriptions of behavior and symptoms with a study of the background of each reaction, yet he follows the standard method of grouping by categories. Psychiatric states are divided into amentia, the organic psychosis, constitutional psychosis, the psychosis associated with other organic diseases, the psychopathic personality, and the psychoneurosis.

Dr. Kraines' discussion of the fundamental psychology of the reaction types follows the idea that the personality is a "function of the socio-psychobiologic unity in a given setting at a given time." This discussion continues through the first four chapters with the conclusion that "the clinical and experimental studies cited above are indicative of the oneness of the organism," which merely seems to be his way of expressing the principle of Meyerian psychobiology.

He does not attempt to discuss the role of the sexual instinct in the preceding chapters but reserves a special chapter entitled "Sex Drives" for this subject. In a most practical manner he takes up the subject of the normal

sex attitude and practice, and then gives a sensible discussion of the psychogenesis of various forms of sexual maladjustment.

The next 220 pages are concerned almost entirely with a description of the methods and the application of the various phychiatric and psychotherapeutic techniques. Again it should be stated that Dr. Kraines handles the psychoneuroses in an understandable fashion, and practical suggestions for management and treatment of these conditions are offered. This is certainly the most profitable portion of the book for the practicing psychiatrist or internist. The other psychopathic states and the psychoses are adequately described as to signs, symptoms, treatment and prognosis.

The last chapter in the book is about psychoanalysis and related schools of psychology. The only criticism of the material is that his forceful method of expression keeps him from dealing very gently with these methods of psychotherapy. He does present an impartial abstract of the text "The Structure and Meaning of Psychoanalysis" by Healy, Bronner and Bowers. The reviewer feels that he has adequately fulfilled his initial precept as indicated in the sub-title, that is, a "socio-psychobiologic analysis and resynthesis," to produce a useful book on therapy.

Hamilton Ford, M.D. Galveston, Texas

A SURGEON'S AUTOBIOGRAPHY, by Hugh Young, M.D., Published by: Harcourt, Brace and Co., 1940.

There is a saying, that no one should write a review who does not love and honor the author; in this case any reader might write it because of the respect he must inescapably feel for Dr. Young.

The author calls his book an autobiography, but it is more than that. The universality of his point of view causes it to grow beyond its frame and become part of medical history. Dr. Hugh Young describes in captivating and humorous manner his youth, with grateful mention of his parents and forebears. His first ambitions were journalistic and his autobiography and his scientific papers prove that he would also have reached mastery in that field. In ample time he found that he was born to become a scholar and a physician, essentially a urologist, and to render great service to suffering men and women. Dr. Young describes the years of his apprenticeship, and introduces the reader to the physician's workshop and recalls his teachers and collaborators.

It would exceed the limits of a review to enumerate all the interesting details of the book; it must be left to the reader to seek out the most significant passages. It is possible the reader will consider them to be the whole book. Notwithstanding, special attention should be called here to some chapters of particular interest. In some of the chapters Dr. Young describes in a simple way intelligible both to the expert and layman the construction and development of surgical instruments needed by the urologist (many invented by him and his collaborators). He also describes new methods used for genitourinary operations, and the utilization of inventions in all the fields related to urology. With regard to these descriptions the book may be almost called a textbook. Two of the most interesting chapters are entitled "Diamond Jim Brady" and the "James Buchanan Brady Urological Institute," not only because of the facts mentioned but because they prove that in America wealthy men have been conscious of their public duties and were anxious to be connected with science, if only as underwriters.

"Diamond Jim Brady," (born James Buchanan Brady) was a poor boy, who eventually became very rich, and submitted to a dangerous operation in view of his cardiac disease. Mr. Brady owed his life to the operation, which had been executed with great ingenuity by Dr. Young. On Dr. Young's suggestion, Mr. Brady gratefully donated a large sum for the foundation of a urological institute. Dr. Young was in charge of the planning and later the management of the institute. Chapter 8 describes how Dr. Young devoted his knowledge, care and time to this foundation, its arrangement and research and how he engaged the best collaborators so that it might raise the standards of research and training in Dr. Young's branch of medicine. During the World War, Dr. Young was active in Europe. Recognizing the weakness of European medical organization, he did much to improve it. He also worked actively with the problems of venereal diseases and other infections. These were exacting as many victims as hostile arms. The chapters describing this part of his life are among the best parts of the autobiography.

After the World War, the author returned to his beloved Maryland. He was called upon to treat President Wilson during his last illness. In spite of his extensive practice, Dr. Young devoted a good part of his life to the welfare of his home state, Maryland, which owes to his energetic mind the legislation for the control of Tuberculosis, the legislation for the care of the insane and the foundation of a municipal hospital in Baltimore. This versatility found medical activity not enough to occupy him but he was active in the fine arts and music. In some chapters he described his travels in other continents, where he was paid high honor.

The book would not be complete without mention of his family life. In his reticence, there are not many pages, but they are of such heartiness, that we feel with him, are happy with him and mourning with him, as his beloved wife was taken early from him. In his deep sorrow he does not forget mankind. Since his wife died from blood-poisoning caused by an apparently harmless toothache, he warns against neglect of dental problems, insignificant as they may appear.

The dignified appearance of the volume corresponds to the high standard of the book.

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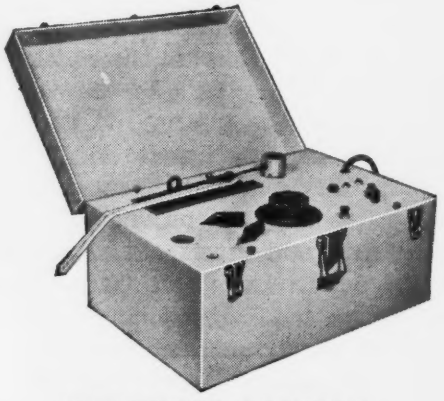

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